PB88-161971

Health and Environmental Effects Profile for Allidochlor

(U.S.) Environmental Protection Agency
Cincinnati, OH

Jan 84

U.S. Department of Commerce
National Technical Information Service
PUBS

PB86-161971

EPA/600/X-84/333
January 1984

HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR ALLIDOCHLOR

ENVIRONMENTAL CRITERIA AND ASSESSMENT OFFICE
OFFICE OF HEALTH AND ENVIRONMENTAL ASSESSMENT
OFFICE OF RESEARCH AND DEVELOPMENT
U.S. ENVIRONMENTAL PROTECTION AGENCY
CINCINNATI, OH 45268

REPRODUCED BY
U.S. DEPARTMENT OF COMMERCE
NATIONAL TECHNICAL
INFORMATION SERVICE
SPRINGFIELD, VA. 22161

TECHNICAL REPORT DATA (Please read Instructions on the reverse before completing)		
1. REPORT NO. EPA/600/X-84/333	2.	3. RECIPIENT'S ACCESSION NO. PB88 161971/AS
4. TITLE AND SUBTITLE Health and Environmental Effects Profile for Allidochlor		5. REPORT DATE January 1984
		6. PERFORMING ORGANIZATION CODE
7. AUTHOR(S)		8. PERFORMING ORGANIZATION REPORT NO.
9. PERFORMING ORGANIZATION NAME AND ADDRESS		10. PROGRAM ELEMENT NO.
		11. CONTRACT/GRANT NO.
12. SPONSORING AGENCY NAME AND ADDRESS Environmental Criteria and Assessment Office Office of Research and Development U.S. Environmental Protection Agency Cincinnati, OH 45268		13. TYPE OF REPORT AND PERIOD COVERED
		14. SPONSORING AGENCY CODE EPA/600/22
15. SUPPLEMENTARY NOTES		

16. ABSTRACT

The Health and Environmental Effects Profile for Allidochlor was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. Existing data are insufficient to determine an Acceptable Daily Intake (ADI) or a carcinogenic potency factor for Allidochlor.

17. KEY WORDS AND DOCUMENT ANALYSIS		
a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
18. DISTRIBUTION STATEMENT Public	19. SECURITY CLASS (This Report) Unclassified	21. NO. OF PAGES 31
	20. SECURITY CLASS (This page) Unclassified	22. PRICE PC A03/MF A01

EPA Form 2220-1 (Rev. 4-77) PREVIOUS EDITION IS OBSOLETE

DISCLAIMER

This document has been reviewed in accordance with U.S. Environmental Protection Agency policy and approved for publication. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched."

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include, Acceptable Daily Intakes (ADIs) for chronic exposures. An ADI is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, ADIs are not estimated in this document series. Instead, a carcinogenic potency factor or q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA docket.

ACKNOWLEDGEMENTS

The initial draft of this report was prepared by Syracuse Research Corporation under Contract No. 68-03-3228 for U.S. EPA's Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. Dr. Christopher DeRosa was the Technical Project Monitor and Helen Ball was the Project Officer. The final documents in this series were prepared for the Office of Solid Waste, Washington, DC.

Scientists from the following U.S. EPA offices provided review comments for this document series:

Environmental Criteria and Assessment Office, Cincinnati, OH
Carcinogen Assessment Group
Exposure Assessment Group
Reproductive Effects Assessment Group

Editorial review for the document series was provided by:
Judith Olsen and Erma Durden
Environmental Criteria and Assessment Office, Cincinnati, OH

Technical support services for this document series were provided by:
Bette Zwayer, Pat Daunt, Karen Mann, and Jacky Bohanon
Environmental Criteria and Assessment Office, Cincinnati, OH.

TABLE OF CONTENTS

		Page
1.	INTRODUCTION	1
1.1.	STRUCTURE AND CAS NUMBER	1
1.2.	PHYSICAL AND CHEMICAL PROPERTIES	1
1.3.	PRODUCTION DATA	2
1.4.	USE DATA	2
2.	ENVIRONMENTAL FATE AND TRANSPORT PROCESSES	4
2.1.	WATER	4
2.2.	AIR	5
2.3.	SOIL	5
3.	EXPOSURE	8
3.1.	WATER	8
3.2.	FOOD	8
3.3.	INHALATION	8
3.4.	DERMAL	8
4.	PHARMACOKINETICS	9
4.1.	ABSORPTION	9
4.2.	DISTRIBUTION	9
4.3.	METABOLISM	9
4.4.	EXCRETION	9
5.	EFFECTS	11
5.1.	CARCINOGENICITY	11
5.2.	MUTAGENICITY	11
5.3.	TERATOGENICITY	11
5.4.	OTHER REPRODUCTIVE EFFECTS	11
5.5.	CHRONIC AND SUBCHRONIC TOXICITY	11
5.6.	OTHER RELEVANT INFORMATION	11
6.	AQUATIC TOXICITY	13
7.	EXISTING GUIDELINES AND STANDARDS	14
7.1.	HUMAN	14
7.2.	AQUATIC	14
8.	RISK ASSESSMENT	15
9.	REFERENCES	16
APPENDIX:	LITERATURE SEARCHED	22

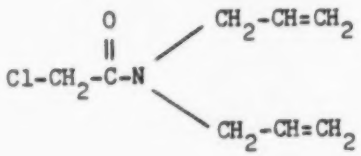
LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
BCF	Bioconcentration factor
bw	Body weight
GI	Gastrointestinal
LD_{50}	Dose lethal to 50% of recipients
NOEL	No-observed-effect level
TLC	Thin layer chromatography
UV	Ultraviolet

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

Allidochlor is the common name for N,N-diallyl-2-chloroacetamide. Allidochlor is commonly referred to as CDAA. Current Chemical Abstracts usage is 2-chloro-N,N-di-2-propenylacetamide. Other chemical synonyms include 2-chloro-N,N-diallylacetamide and N,N-diallyl-α-chloroacetamide. Allidochlor was introduced by the Monsanto Company under code number CP6343 and trade mark "Randox" (BCPC, 1977). Other trade marks or names include "Radox" and "NCI-C04035". The structural and molecular formulae for allidochlor are as follows:



Empirical formula: $C_8H_{12}ClNO$

Molecular weight: 173.6

The Chemical Abstracts Service (CAS) Registry number is 93-71-0.

1.2. PHYSICAL AND CHEMICAL PROPERTIES

Allidochlor is an oily, amber liquid at room temperature (WSSA, 1979). Selected physical properties are listed below:

Boiling point:	74°C at 0.3 mm Hg (WSSA, 1979) 92°C at 2.0 mm Hg (BCPC, 1977)
Vapor pressure:	9.4×10^{-3} mm Hg at 20°C (WSSA, 1979)
Decomposition temperature:	125°C (WSSA, 1979)
Refractive index, n^{25}:	1.4932 (BCPC, 1977)

Water solubility: 19,700 ppm (Kenaga, 1980)	2% at 25°C (WSSA, 1979) 1.97 g/100 ml at 25°C (BCPC, 1977)
BCF: (calculated from water solubility)	2 (Kenaga, 1980)
Soil sorption coefficient: (calculated from water solubility)	19 (Kenaga, 1980)

Allidochlor is moderately soluble in petroleum hydrocarbons and is readily soluble in ethanol and xylene (BCPC, 1977). Chemically, the chlorine atom is unstable (Spencer, 1968). Allidochlor has relatively good resistance to decomposition by UV irradiation (WSSA, 1979).

1.3. PRODUCTION DATA

ALlidochlor is made by the reaction of diallylamine with chloroacetyl chloride (BCPC, 1977; Spencer, 1968).

The only domestic manufacturer of allidochlor is Monsanto in Muscatine, IA (SRI, 1983; USITC, 1983). Current production figures are not available; however, production in 1971 was reportedly 10 million pounds (Ouellette and King, 1977).

1.4. USE DATA

Allidochlor is a selective pre-emergence herbicide used to control most annual grasses and certain broadleaf weeds (WSSA, 1979; BCPC, 1977). It can also be used post-emergence on onions and sugarcane. Rates of application are 4-5 pounds/acre for most crops and 10 pounds/acre for sugarcane.

In 1975, domestic consumption of allidochlor totalled 6.3 million pounds with 0.2 million pounds used in corn, 0.1 million pounds used in vegetables and 6.0 million pounds used in other field crops (SRI, 1976). In 1979, 187,000 pounds were consumed in onion fields in New York State (Ferguson and

McCalla, 1981a), 71,000 pounds were consumed in onion fields in Michigan, Minnesota and Ohio, 29,500 pounds in corn fields in the Midwest (Webb and Ferguson, 1981) and 3800 pounds were used in celery fields in Florida (Ferguson and McCalla, 1981b); this is probably only part of allidochlor consumption for 1979.

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

2.1. WATER

There are few data regarding the environmental fate and transport of allidochlor in water. Like other chemicals, the fate of allidochlor in water will depend on its ability to undergo chemical, photochemical and biological reactions. While the hydrolysis of allidochlor has not been studied, the hydrolysis of the chemically related chloroacetamide has been (Mabey and Mill, 1978). At 25°C and pH 7, the hydrolysis half-life of chloroacetamide is 1.46 years. Mabey and Mill (1978) have noted that alkyl groups on the nitrogen retard both acid and based catalyzed hydrolysis processes on amides; therefore, it is likely that the hydrolysis half-life of allidochlor is on the same order of magnitude as chloroacetamide. With a hydrolysis half-life of ~1.5 years, the hydrolysis of allidochlor is not likely to be environmentally significant.

The UV spectra of allidochlor showes zero absorption above 260 nm (Gore et al., 1971). Therefore, direct photolysis of allidochlor in the environment will produce no decomposition. The possibility of photosensitized photodegradation has not been investigated.

Microbial degradation of allidochlor in the aquatic media has not been studied; however, soil microorganisms have demonstrated an ability to biodegrade allidochlor (Section 2.3.) and, therefore, it is possible that allidochlor is biodegraded in the water (Kaufman and Blake, 1973).

The transport of allidochlor from water via volatilization has not been studied; however, Deming (1963) studied its volatilization from soil surfaces. WSSA (1979) reported that in the absence of soil adsorptive forces, 4 pounds/acre of allidochlor volatilizes in <8 hours at $\geq$7°C. Deming (1963) has noted that increasing amounts of water in the soil

accelerates volatilization loss of allidochlor. Therefore, it is possible that the volatilization transport of allidochlor is a predominant mechanism by which it is lost from water.

Kenaga (1980) has predicted a BCF of 2 for allidochlor, based upon its water solubility. The author stated that BCF values <100 indicate that the compound is unlikely to be bioconcentrated.

The persistence of allidochlor in water has not been studied and is difficult to predict from the available data.

2.2. AIR

Data specific to the fate and transport of allidochlor in the atmosphere were not located in the available literature. Direct photolysis of allidochlor in the air is not expected to occur since the UV spectra of allidochlor shows zero absorption above 260 nm (Gore et al., 1971).

2.3. SOIL

The fate and transport of allidochlor in soil has been more adequately studied than its fate and transport in the air or in water. WSSA (1979) has reported that microbial breakdown is primarily responsible for the decomposition of allidochlor in soil, while hydrolysis is secondary. Several microorganisms have been shown to biodegrade allidochlor. Pseudomonas striata and an Achromobacter species were able to degrade allidochlor after isolation from soil (Kaufman and Blake, 1973).

Direct photolysis of allidochlor at the soil surface is not expected to occur since the UV spectrum of allidochlor shows zero absorption above 260 nm (Gore et al., 1971). The potential for sensitized photodegradation, with sensitizers such as humic acids, has not been investigated.

The transport of allidochlor from soil due to volatilization has been studied (Deming, 1963). WSSA (1979) reported that, in the absence of

adsorption, 4 pounds/acre of allidochlor volatilizes in <8 hours at ≥7°C. In the presence of adsorption, a virtually ever present environmental condition, the volatilization rate will decrease. Deming (1963) found the rate of volatilization of allidochlor to increase with increasing amounts of water in the soil and with decreasing organic matter content of soil. Increasing temperature usually increased volatilization, although occasionally it decreased. When surface-applied allidochlor is incorporated into the soil, by means such as discing or harrowing, the volatilization rate can be significantly retarded (Sheets, 1959; WSSA, 1979). Therefore, the overall loss of allidochlor from soil due to volatilization is affected by moisture content, organic matter content and incorporation practices (or lack of them). In most instances, the volatilization loss of allidochlor from soils is significant and an important removal mechanism.

Leaching is related to adsorption, which has already been shown to be related to volatilization. Adsorption of allidochlor is directly proportional to the colloid content of soil (WSSA, 1979). Therefore, the content of the soil will be a determining factor in the leachability of applied allidochlor. For example, Upchurch (1966) has noted that allidochlor is tightly bound at dry mineral soil surfaces but easily leached with high moisture content for potential volatilization. Leaching will be reduced in soils that contain with high levels of organic matter. Helling et al. (1971) have determined the mobility of allidochlor in Hagerstown silty clay loam by means of soil TLC (R_f of 0.82). The R_f value of 0.82 indicates that allidochlor is mobile in soil. The predicted soil sorption coefficient (K_{oc}) of 19 for allidochlor (Kenaga, 1980), based upon its water solubility, indicates that allidochlor is moderately to highly mobile in soil. By

contrast, Weber and Best (1972), in an abstract of the proceedings of a meeting, reported that allidochlor was relatively immobile in a North Carolina soil.

The persistence of allidochlor in soil has been studied by several investigators. Sheets (1959) treated eight California soils with 2.5-160 ppm allidochlor in a greenhouse; using an oat bioassay, he determined that the allidochlor was degraded to innocuous levels within 3 months. Gantz (1960) applied allidochlor (8 pounds/acre) to soil and found that it presists for 4 weeks in moist soils and somewhat longer in dry soil; also, persistence was greater in soil at 80 or 100°F than at 40 or 60°F. Otten et al. (1957) found allidochlor persistence to decrease in moist soils with increasing temperatures. In a review of pesticide persistence, Kearney et al. (1969) listed the persistence of allidochlor as 2 months. Using an aerobic incubation system in the laboratory, Marvel et al. (1978) found allidochlor to have a soil half-life of 0.2 weeks with only 6.7% of the allidochlor undegraded after 2 weeks. WSSA (1979) has reported that the resultant average persistence of allidochlor at recommended application rates is 3-6 weeks, with shorter persistence on soils of low colloid content.

3. EXPOSURE

3.1. WATER

The U.S. EPA STORET data base contains no listings or data for allidochlor. Other monitoring data for allidochlor in water were not located in the available literature.

3.2. FOOD

Monitoring data for allidochlor exposure in food were not located in the available literature. WSSA (1979) reports that allidochlor is rapidly metabolized in plants, and the degradation products are glycolic acid and an amine.

3.3. INHALATION

Monitoring data for allidochlor exposure in the air were not located in the available literature. As noted in Section 2.3., allidochlor can volatilize quite rapidly from soil surfaces. This indicates the possibility of inhalation exposure to workers in the field to which allidochlor has recently been applied.

3.4. DERMAL

Monitoring data for allidochlor exposure on the skin of American workers were not located in the available literature. Peachey (1981) has reported that allidochlor has caused contact dermatitis in the United Kingdom.

4. PHARMACOKINETICS

4.1. ABSORPTION

As described in Section 4.4., the recovery of 80-90% of the radioactivity from the urine of rats 24 hours after a single oral dose (22 mg/kg) of the labelled allidochlor indicates that allidochlor is readily absorbed from the GI tract. In the same experiment, <3% of the recovered label was found in the feces after 48 hours (Lamoureux and Davison, 1975).

4.2. DISTRIBUTION

Pertinent data regarding the distribution of allidochlor were not located in the available literature.

4.3. METABOLISM

Mercapturic acid accounted for 89% of the label recovered in the urine 24 hours after rats received a single dose (22 mg/kg bw) of ^{14}C-carbonyl-labeled allidochlor. The major metabolite was identified as the sulfur substituted dichlorination product of allidochlor. In addition, allidochlor formed apparent glutathione conjugates when incubated in vitro with a crude rat liver homogenate (Lamoureux and Davison, 1975). Allidochlor remained stable for 24 hours when incubated with filtered bovine rumen fluid; however, it was not detectable after 30 minutes of incubation with fractionated (10,000 xg supernatant) beef liver (St. John and Lisk, 1974).

4.4. EXCRETION

Two male Sprague-Dawley rats received single oral doses (22 mg/kg bw) of ^{14}C-carbonyl-labeled allidochlor by intubation. Measurement of the ^{14}C in the urine, feces, CO_2 and carcass indicated that 80-90% of the ^{14}C was recovered in the urine after 24 hours. An additional 1.3-1.4% of the labeled C was recovered in the urine after 48 hours. Eighty-nine percent of the ^{14}C label recovered in the urine after 24 hours was present as the

corresponding mercapturic acid. Without undergoing any other chemical transformations, the allidochlor molecule substituted glutathione in place of its chloride. Small percents of the radioactive allidochlor were recovered in the feces (0.17-1.5% after 24 hours; 1.1-2.4% after 48 hours), the expired air (0.04%) and the animal carcass (1.2-2.6% after 48 hours). The total amount of radioactive label recovered was 87-95% (Lamoureux and Davison, 1975). Without reporting any methodology, Miaullis et al. (1978) stated that 4 days after rats were given a single oral dose of ^{14}C-allidochlor, the percentage of radioactive label recovered from the urine was 73%; the feces, 13%; the expired CO_2, 5%; and the animal carcasses, 6%. St. John and Lisk (1974) found no allidochlor in the milk, urine or feces of one Holstein cow fed allidochlor (0.2374 mg/kg bw/day) for 4 days. The expired CO_2 was not studied. No attempts were made to identify metabolites of allidochlor in the milk, urine or feces (St. John and Lisk, 1974).

5. EFFECTS

5.1. CARCINOGENICITY

Pertinent data regarding the carcinogenicity of allidochlor were not located in the available literature.

5.2. MUTAGENICITY

Allidochlor was reported to be nonmutagenic to eight unspecified strains of Salmonella typhimurium, assayed for reverse mutation in the spot test (Andersen et al., 1972). The zones of inhibition that were observed, indicated that sufficient allidochlor diffused into the medium. Allidochlor was assayed for forward mutations (to rII type mutants) in bacteriophage T_4 and reverse mutation in two rII mutants (AP72 and N17) of bacteriophage T_4 (Andersen et al., 1972), and negative results were obtained. Since mammalian metabolic activating sytem (S-9) was not used in any of these assays, they have limited capability for hazard evaluation.

5.3. TERATOGENICITY

Pertinent data regarding the teratogenicity of allidochlor were not located in the available literature.

5.4. OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding the reproductive effects of allidochlor were not located in the available literature.

5.5. CHRONIC AND SUBCHRONIC TOXICITY

Pertinent data regarding the chronic and subchronic toxicity of allidochlor were not located in the available literature.

5.6. OTHER RELEVANT INFORMATION

Allidochlor has been reported to cause contact dermatitis (Spencer, 1966) in humans. The only effect seen in one sheep after oral administration of 25 mg/kg/day of allidochlor for 10 days was slight or negligible

weight loss. A higher dose (250 mg/kg bw) given orally for 1 or 2 days "poisoned" one sheep (Palmer, 1964). The LD_{50} in rats of allidochlor is 700 mg/kg for oral administration (Bailey and White, 1965) and 360 mg/kg for topical administration (Wiswesser, 1976). No effect was seen in rats given 70 mg/kg of allidochlor for 30 days (BCPC, 1977).

6. AQUATIC TOXICITY

The toxicity of allidochlor to aquatic organisms has been tested in a limited number of studies. Apparently, allidochlor was tested in several Japanese studies by Nishiuchi (1977, 1980) and Nishiuchi and Asano (1979) in fish (Medaka, Orysins latipes; carp, Cyprinus carpio), an amphibian (frog, Bufo vulgaris) and an insect (Cloeon dipterum). Toxicity data or median lethal levels of allidochlor in these species were not reported in the English abstracts of the Japanese studies and translated copies of these publications were not available for review.

Allidochlor was toxic to the green alga, Chlorella vulgaris, at 10 mg/ℓ, causing at least 50% growth inhibition within 24 hours (Kratky and Warren, 1971). At 1 mg/ℓ, allidochlor did not cause a 50% inhibition of growth during the 24-hour test.

Additional data regarding the toxic effects of allidochlor to aquatic organisms following acute or chronic exposure or data on residues were not located in the available literature.

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

In 1970, the FDA established a tolerance of 0.05 ppm for allidochlor on raw agricultural commodities (Federal Register, 1970). Allidochlor, if applied before the emergence, of corn is exempt from tolerance when used as an inert ingredient of s-ethyldiisobutylthiocarbamate, s-ethyl dipropylthiocarbamate or s-propyl dipropylthiocarbamate formulations (Code of Federal Regulations, 1981).

7.2. AQUATIC

Guidelines and standards for the protection of aquatic organisms from the toxic effects of allidochlor were not located in the available literature.

8. RISK ASSESSMENT

Pertinent data regarding carcinogenicity, teratogenicity or other reproductive effects of allidochlor were not located in the available literature.

Experimental evidence from the rat indicates that allidochlor is absorbed quickly from the GI tract, conjugated to glutathione and excreted in the urine within 24 hours (Lamoureux and Davison, 1975). Although a dose of 70 mg/kg allidochlor for 30 days in rats is a NOEL (BCPC, 1977) and a dose of 25 mg/kg for 10 days in sheep produced slight or negligible weight loss (Palmer, 1964), there are insufficient data to establish an ADI.

Statements concerning available literature in this document refer to published, quotable sources and are in no way meant to imply that confidential data, which this document could not address, are not in existence. These data, which are considered Confidential Business Information (CBI), are available in summary form as a CBI Appendix that can be accessed by individuals with appropriate clearance. Conclusions from the CBI Appendix regarding risk assessment can, however, be presented in this chapter.

Several animal studies of the effects of allidochlor exposure were available for review in the CBI Appendix to this document. These studies were classified as invalid (U.S. EPA, 1983); therefore, data remain insufficient for the derivation of an ADI or a q_1^*.

9. REFERENCES

Andersen, K.J., E.G. Leighty and M.T. Takahashi. 1972. Evaluation of herbicides for possible mutagenic properties. J. Agric. Food Chem. 20: 649-656.

Bailey, G.W. and J.L. White. 1965. Herbicides: A compilation of their physical, chemical and biological properties. Residue Rev. 10: 97-122.

BCPC (British Crop Protection Council). 1977. Allidochlor. In: Pesticide Manual, 5th ed., H. Martin and C.R. Worthing, Ed. BCPC Publications, Worcestershire, England. p. 8.

Code of Federal Regulations. 1981. N,N-Diallyl dichloroacetamide; exemption from requirement of a tolerance. 40 CFR 180.1026.

Deming, J.M. 1963. Determination of volatility losses of CDAA-C^{14} from soil surfaces. Weeds. 11: 91-96. (CA 59: 3268h)

Federal Register. 1970. Tolerances and exemptions from tolerances for pesticide chemicals in or on raw agricultural commodities. 2-Chloro-N,N-diallylacetamide. Federal Register. 35(112): 8930.

Ferguson, W.L. and I.E. McCalla. 1981a. 1979 Pesticide Use on Vegetables in the Northeast, A Preliminary Report. ERS Staff Report No. AGES911218. Natural Resource Economics Div., Economic Research Serv., U.S. Dept. of Agriculture, Washington, DC.

Ferguson, W.L. and I.E. McCalla. 1981b. 1979 Pesticide Use on Florida Vegetables, A Preliminary Report. ERS Staff Report No. AGES911218. Natural Resource Economics Div., Economic Research Serv., U.S. Dept. of Agriculture, Washington, DC.

Gantz, R.L. and F.W. Slife. 1960. Persistence and movement of CDAA and CDEC in soil and the tolerance of corn seedlings to these herbicides. Weeds. 8: 599-606. (Cited in Sanborn et al., 1977)

Gore, R.C., R.W. Hannah, S.C. Pattacini and T.J. Porro. 1971. Infrared and ultraviolet spectra of senventy-six pesticides. J. Assoc. Offic. Anal. Chem. 54(5): 1040-1082.

Helling, C.S., D.D. Kaufman and C.T. Dieter. 1971. Algae bioassay detection of pesticide mobility in soils. Weed Sci. 19(6): 685-690.

Kaufman, D.D. and J. Blake. 1973. Microbial degradation of several acetamide, acylanilide, carbamate, toluidine and urea pesticides. Soil Biol. Biochem. 5: 297-308.

Kearney, P.C., E.A. Woolson, J.R. Plimmer and A.R. Isensee. 1969. Decontamination of pesticides in soil. Residue Rev. 29: 137.

Kenaga, E.E. 1980. Predicted bioconcentration factors and soil sorption coefficients of pesticides and other chemicals. Ecotoxicol. Environ. Safety. 4: 26-38.

Kratky, B.A. and G.F. Warren. 1971. Use of three simple, rapid bioassays on forty-two herbicides. Weed Res. 11(4): 257-262.

Lamoureux, G.L. and K.L. Davison. 1975. Mercapturic acid formation in the metabolism of propachlor, CDAA and fluorodifen in the rat. Pestic. Biochem. Physiol. 5(6): 497-506.

Mabey, W. and T. Mill. 1978. Critical review of hydrolysis of organic compounds in water under environmental conditions. J. Phys. Chem. Ref. Data. 7(2): 413.

Marvel, J.T., B.B. Brightwell, J.M. Malik, M.L. Sutherland and M.L. Rueppel. 1978. A simple apparatus and quantitative method for determining the persistence of pesticides in soil. J. Agric. Food Chem. 26(5): 1116-1120.

Miaullis, J.B., V.M. Thomas, R.A. Gray, J.J. Murphy and R.M. Hollingworth. 1978. Metabolism of R-25788 (N,N-diallyl-2,2-dichloroacetamide) in Corn Plants, Rats and Soil. Chem. Action Herbic. Antidotes. (Symp.) F.M. Pallos and J.E. Casida, Ed. Academic Press, New York, NY. p. 109-131.

Nishiuchi, Y. 1977. Toxicity of formulated pesticides to some freshwater organisms. XXXX. Suisan Zoshoku. 24(4): 140-145. (Jap.)

Nishiuchi, Y. 1980. Effect of pH on the toxicity of pesticides to aquatic animals. II. Seitai Kagaku. 3(2): 73-79. (Jap.)

Nishiuchi, Y. and K. Asano. 1979. Toxicity of pesticides to some freshwater organisms. LIX. Suisan Zoshoku. 27(1): 48-55. (Jap.)

Otten, R.J., J.E. Dawson and M.M. Schreiber. 1957. Persistence and leaching of 2-chloroallyl diethyldithiocarbamate (CDEC) and α-chloro-N,N-diallylacetamide (CDAA) in soil. Proc. Northeast Weed Contr. Conf. 1957: 111-119. (Cited in Sanborn et al., 1977)

Ouellette, R.P. and J.A. King. 1977. Chemical Week Pesticides Register. McGraw-Hill Book Co., NY. p. 265.

Palmer, J.S. 1964. Toxicity of carbamate, triazine, dichloropropionanilide, and diallylacetamide compounds to sheep. J. Am. Vet. Med. Assoc. 145(9): 917-920.

Peachey, R.D.G. 1981. Skin hazards in farming. Br. J. Dermatol. 105(21): 45-50. (Taken from Pestab/81/3472.)

Sanborn, J.R., R.L. Metcalf and B.M. Francis. 1977. The Degradation of Selected Pesticides in Soil: A Review of the Published Literature. U.S. EPA, Cincinnati, OH. EPA-600/9-77-022, NTIS PB 272 353.

Sheets, T.J. 1959. Effects of soil type and time on the herbicidal activity of CDAA, CDEC, and EPTC. Weeds. 7: 442-448.

Spencer, M.C. 1966. No title given. J. Am. Med. Assoc. 198: 1307. (Taken from Br. J. Dermatol. 1981) 105(Supl. 21): 50.

Spencer, E.Y. 1968. Guide to the Chemicals Used in Crop Protection, 5th ed. Publ. 1093, Canada Department of Agriculture, Research Branch.

SRI (Stanford Research Institute). 1976. Herbicides. In: Chemical Economics Handbook. Stanford Research Institute, Menlo Park, CA. p. 573.7008E-F.

SRI (Stanford Research Institute). 1983. 1983 Directory of Chemical Producers: United States of America. SRI International, Menlo Park, CA. p. 775.

St. John, L.E., Jr. and D.J. Lisk. 1974. Feeding studies with Randox in the dairy cow. Bull. Environ. Contam. Toxicol. 11(6): 529-531.

Upchurch, R.P. 1966. Behavior of herbicides in soil. Residue Rev. 16: 46-85.

U.S. EPA. 1983. Summary of the IBT Review Program, Office of Pesticide Programs, U.S. EPA, Washington, DC. July 1983.

USITC (United States International Trade Commission). 1983. Synthetic Organic Chemicals. U.S. Production and Sales, 1982. USITC Publ. 1422. U.S. International Trade Commission. p. 233.

Webb, S-E.H. and W.L. Ferguson. 1981. 1979 Pesticide Use on Vegetables in the Midwest, A Preliminary Report. ERS Staff Report No. AGES811217. Natural Resource Economics Division, Economic Research Service, U.S. Department of Agriculture, Washington, DC.

Weber, J.B. and J.A. Best. 1972. Activity and movement of 13 soil-applied herbicides as influenced by soil reaction. Proc. S. Weed Sci. Soc. 25: 403-413. (CA 77: 110456a)

Wiswesser, W.J., Ed. 1976. Pesticide Index. Published by College Park: The Entomological Society of America. p. 7.

WSSA (Weed Science Society of America). 1979. CDAA. In: Herbicide Handbook of the Weed Science Society of America, 4th ed. WSSA, Champaign, IL. p. 92-95.

APPENDIX:
LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of:

CA SEARCH (Files 308, 309, 310, 311, 320)
TOXLINE
MEDLINE
RTECS
SCI SEARCH
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE

These searches were conducted in November, 1983. In addition, hand searches were made of Chemical Abstracts (Collective Indices 6 and 7th), and the following secondary sources were reviewed:

ACGIH (American Conference of Governmental Industrial Hygienists). 1980. Documentation of the Threshold Limit Values, 4th ed. (Includes Supplemental Documentation, 1981, 1982, 1983). Cincinnati, OH. p. 486.

ACGIH (American Conference of Governmental Industrial Hygienists). 1983. TLVs: Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1983-84. Cincinnati, OH. p. 94.

BCPC (British Crop Protection Council). 1977. Pesticide Manual, 5th ed., Martin, H. and C.R. Worthing, Ed. British Crop Protection Council. p. 593.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2A. John Wiley and Sons, NY. p. 2878.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2B. John Wiley and Sons, NY. p. 2879-3816.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2C. John Wiley and Sons, NY. p. 3817-5112.

Grayson, M. and D. Eckroth, Ed. 1978-83. Kirk-Othmer Encyclopedia of Chemical Technology, 3rd ed. John Wiley and Sons, NY. 23 Volumes.

Hamilton, A. and H.L. Hardy. 1974. Industrial Toxicology, 3rd ed. Publishing Sciences Group, Inc., MA. p. 575.

ITII (International Technical Information Institute). 1982. Toxic and Hazardous Industrial Chemicals Safety Manual for Handlingand Disposal with Toxicity and Hazard Data. ITII, Tokyo, Japan. p. 700.

NTP (National Toxicology Program). 1983. Carcinogenesis Testing Program. Chemicals on Standard Protocol. Management Status.

Ouellette, R.P. and J.A. King. 1977. Chemical Week Pesticide Register. McGraw-Hill Book Co., NY.

Sax, I.N. 1979. Dangerous Properties of Industrial Materials, 5th ed. Van Nostrand Reinhold Co., NY.

SRI (Stanford Research Institute). 1983. Directory of Chemical Producers. Stanford, CA.

U.S. EPA. 1982. Chemical Activities Status Report, 3rd ed. (EPACASR). Offices of Pesticides and Toxic Substances, Washington, DC. EPA 560/TIIS-82-002b.

U.S. EPA. 1983. Status Report on Rebuttable Presumption Against Registration (RPAR) or Special Review Process. Registration Standards and the Data Call In Programs. Office of Pesticide Programs, Washington, DC.

U.S. EPA. 1983. CHIB Existiny Chemical Assessment Tracking System. Name and CAS Number Ordered Indexes. Office of Toxic Substances, Washington, DC.

USITC (United States International Trade Commission). 1983. Synthetic Organic Chemicals. U.S. Production and Sales, 1982. USITC Publication 1422. Washington, DC.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals, 2nd ed. Van Nostrand Reinhold Co., NY.